**110**

GA- FL258
NR- 102
TI- CARDIAC ELECTROPHYSIOLOGY OF ADENOSINE - BASIC AND CLINICAL CONCEPTS
AU- LERMAN BB BELARDINELLI L
CS- CORNELL UNIV,MED CTR,NEW YORK HOSP,DEPT CARDIOL,525 E 68TH ST,STARR 4/NEW YORK//NY/10021 UNIV FLORIDA,COLL MED,DEPT MED,DIV CARDIOL/GAINESVILLE//FL/32611
SO- <JN> CIRCULATION
SO- <PY> 1991
SO- <VO> 83
SO- <IS> N5
SO- <PG> 1499-1509
LA- ENGLISH
DT- REVIEW

**111**

GA- F0732
NR- 218
TI- MINIMAL RESIDUAL NEOPLASTIC DISEASE - CONCEPT, PATHOGENESIS, AND SUPPLEMENTARY THERAPEUTIC POSSIBILITIES
AU- PRINDULL G
CS- UNIV GOTTINGEN,DEPT PEDIAT,HUMBOLDTALEE 38/D-3400 GOTTINGEN//FED REP GER/
SO- <JN> CANCER TREATMENT REVIEWS
SO- <PY> 1986
SO- <VO> 13
SO- <IS> N3
SO- <PG> 177-194
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798091

**112**

GA- TH794
NR- 160
TI- THE SURPRISING HEART - A REVIEW OF RECENT PROGRESS IN CARDIAC ELECTRO-PHYSIOLOGY
AU- NOBLE D
CS- UNIV OXFORD,PHYSIOL LAB/OXFORD OX1 3PT//ENGLAND/
SO- <JN> JOURNAL OF PHYSIOLOGY-LONDON
SO- <PY> 1984
SO- <VO> 353
SO- <IS> AUG
SO- <PG> 1-50
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

**113**

GA- TC392
NR- 94
TI- ELECTROPHYSIOLOGIC EFFECTS OF ADENOSINE-TRIPHOSPHATE AND ADENOSINE ON THE MAMMALIAN HEART - CLINICAL AND EXPERIMENTAL ASPECTS
AU- BELHASSEN B PELLEG A
CS- HAHNEMANN UNIV,LIKOFF CARDIOVASC INST,CARDIAC ELECTROPHYSIOL LAB/PHILADELPHIA//PA/19102 ICHILOV HOSP,TEL AVIV MED CTR,CARDIAC ELECTRO-PHYSIOL LABS/IL-64239 TEL AVIV//ISRAEL/ LANKENAU MED RES CTR,DIV CARDIOL/PHILADELPHIA//PA/00000
SO- <JN> JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
SO- <PY> 1984
SO- <VO> 4
SO- <IS> N2
SO- <PG> 414-424
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

**114**

GA- SR783
NR- 76
TI- MECHANICAL-PROPERTIES OF CELLULAR CARDIAC PREPARATIONS
AU- DECLERCK NM BRUTSAERT DL
CS- UNIV ANTWERP,DEPT PHYSIOL & MED/ANTWERP//BELGIUM/
SO- <JN> PHARMACOLOGY & THERAPEUTICS
SO- <PY> 1984
SO- <VO> 24
SO- <IS> N1
SO- <PG> 133-146
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

**115**

GA- SP419
NR- 264
TI- CEREBRAL-CIRCULATION AND METABOLISM
AU- SIESJO BK
CS- UNIV LUND,EXPTL BRAIN RES LAB/S-22101 LUND//SWEDEN/
SO- <JN> JOURNAL OF NEUROSURGERY
SO- <PY> 1984
SO- <VO> 60
SO- <IS> N5
SO- <PG> 883-908
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

**116**

GA- RX181
NR- 234
TI- EVOKED-RESPONSES IN NORMAL AND DISEASED MUSCLE WITH PARTICULAR REFERENCE TO TWITCH POTENTIATION
AU- KRARUP C
CS- NINCDS,BLDG 10,ROOM 5N230/BETHESDA//MD/20205
SO- <JN> ACTA NEUROLOGICA SCANDINAVICA
SO- <PY> 1983
SO- <VO> 68
SO- <IS> N5
SO- <PG> 269-315
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798093

**117**

GA- PZ022
NR- 123
TI- PHYSIOLOGY OF CALCIUM CURRENT IN CARDIAC-MUSCLE
AU- KEUNG ECH ARONSON RS
CS- YESHIVA UNIV ALBERT EINSTEIN COLL MED,DEPT MED,DIV CARDIOL/BRONX//NY/10461
SO- <JN> PROGRESS IN CARDIOVASCULAR DISEASES
SO- <PY> 1983
SO- <VO> 25
SO- <IS> N4
SO- <PG> 279-296
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

## THEOPHYLLINE, EEG/EP/ERP ETC & HEALTHY/NORMAL VOLS/SUBJECTS
**FROM psycINFO {DIALOG: FILE 11}**

**118**

AN- 00851144
TI- Effects of single and repeated doses of theophylline on aspects of performance, electrophysiology and subjective assessments in healthy human subjects.
AU- Bartel, Peter Delport, Rhena Lotz, Barend Ubbink, Johan et al
CS- HF Verwoerd Hosp, Neuropsychopharmacology Lab, Pretoria, South Africa
SO- <JN> Psychopharmacology
SO- 1992 Jan Vol 106(1) 90-96
SN- 00333158
LA- English
DT- JOURNAL ARTICLE
AB- Evaluated the effects of both single (400 mg) and repeated doses (300 mg for 4 wks) of theophylline on a battery of 9 performance tests, the EEG, the electromyogram (EMG), and on subjective assessments of mood and side effects in 20 healthy adults (aged 20-36 yrs). An information processing test revealed enhanced performance in both phases, while an addition test showed improved performance in the single dose phase only. The remaining 7 performance tests failed to show significant differences between theophylline and placebo. Single doses of theophylline did not significantly alter mood, but marked adverse effects were encountered in the repeated dose phase, possibly related to unpleasant side effects. Findings demonstrate central nervous system (CNS) stimulation by both single and repeated doses of theophylline with the occurrence of adverse side effects during repeated administrations.

**FROM EMBASE** {*Excerpta Medica*; DIALOG: File 73}

**119**

AN- <DIALOG> 7577260
TI- Electrophysiological indices of central and peripheral nervous system function during theophylline therapy
AU- Bartel P. Lotz B. Delport R. Ubbink J. Becker P.
CS- Department of Neurology, University of Pretoria, Pretoria
CS- South Africa
SO- <JN> NEUROPSYCHOBIOLOGY
CP- Switzerland
SO- <PY> 1989
SO- <VO> 21/2 (104-108)
SN- 0302-282X
LA- English
AB- Electrophysiological parameters including the EEG, somatosensory evoked potentials (SEP), F waves, long loop reflexes and peripheral nerve conduction velocities were assessed during the 15th week of theophylline therapy, with serum levels maintained in the low therapeutic range, in young, healthy male volunteers (n = 7). Recordings were repeated 1 week after vitamin B6 supplementation and finally 6-7 weeks after the cessation of medication. Alpha amplitudes were significantly lower during theophylline therapy compared to the posttreatment baseline recording. The SEP findings failed to reveal any significant differences between the three recordings, as was the case with peripheral nerve conduction velocities and long loop reflexes. The average F wave latencies during theophylline therapy were significantly shorter, and the percentage F waves recorded was higher, compared to the baseline recordings. These findings suggest that relatively long-term theophylline therapy has stimulatory effects on aspects of electrical activity in the brain and spinal cord. These effects appeared to be unchanged after vitamin B6 supplementation for 1 week.

2050798096

**120**

AN- <DIALOG> 7140022
TI- Rectum has abnormal ion transport but normal cAMP-binding proteins in cystic fibrosis
AU- Goldstein J.L. Nash N.T. Al-Bazzaz F. Layden T.J. Rao M.C.
CS- Department of Medicine, University of Illinois College of Medicine at Chicago, Chicago, IL 60612
CS- USA
SO- <JN> AM. J. PHYSIOL., CELL PHYSIOL.
CP- USA
SO- <PY> 1988
SO- <VO> 254/5 (23/5) (C719-C724)
SN- 0002-9513
LA- English
AB- The luminal membranes of involved tissues in cystic fibrosis (CF) are relatively impermeable to Cl and the regulation of Cl transport by adenosine 3',5'-cyclic monophosphate (cAMP)-mediated hormones is abnormal. We investigated the human rectum as a putative model for CF. We compared in vivo transrectal potential difference (PD) in CF and in normal subjects in response to sequential perfusions with various test solutions. The base-line PD was different in normal (-35.5 plus or minus 4.0 mV; lumen negative; mean plus or minus SE; n = 9) and CF subjects (-23.4 plus or minus 3.1 mV; n = 6; $P < 0.025$) and was eliminated by amiloride (10-4 M) perfusion in both groups by 3 min. However, in response to a Cl-free solution with amiloride, all six CF subjects exhibit less of a change in PD (PD, -2.2 plus or minus 1.2 mV vs. -11.7 plus or minus 1.5 mV in 6 controls; $P < 0.01$). Furthermore, normal subjects (n = 7) respond to a 5 mM theophylline + amiloride perfusion with an increase in lumen negative PD, whereas CF subjects (n = 6) show no increase in lumen-negative PD. Rectal biopsy specimens from four normal and four CF subjects exhibit similar (2- to 3-fold) increases in theophylline-induced cAMP content and have similar cAMP-binding proteins (CF, n = 3; control, n = 3). We conclude that the rectum is an involved epithelium in CF in which the aberration may lie at a point beyond the binding of cAMP to its protein kinase.

2050798097

**121**

AN- <DIALOG> 6029024
TI- Breathing during sleep
AU- Flenley D.C.
CS- Department of Respiratory Medicine, University of Edinburgh, City Hospital, Edinburgh EH10 5SB
CS- UNITED KINGDOM
SO- <JN> ANN. ACAD. MED. SINGAPORE
CP- SINGAPORE
SO- <PY> 1985
SO- <VO> 14/3 (479-484)
LA- ENGLISH
AB- Hypoxemia during the rapid eye movement phase of sleep is common in 1) older healthy normal subjects over 55 years of age; 2) the sleep apnoea syndromes - such as obstructive sleep apnoea, where oro-nasal airflow ceases for more than 10 seconds on many separate occasions throughout the night, due to failure of contraction of the genio-glossus muscle; 3) 'blue and bloated' patients with chronic bronchitis and emphysema, where profound nocturnal hypoxemia is common in REM sleep, and is associated with further elevation of pulmonary arterial pressure; 4) the overlap syndrome - where 'blue and bloated' chronic bronchitis is associated with an obstructive sleep apnoea syndrome; and 5) bronchial asthma, where hypoxemia is associated with irregular breathing and possibly nocturnal bronchoconstriction. Although absolute recognition depends upon all night sleep studies, monitoring of ear oxygen saturation, breathing patterns, and EEG, the clinical features when awake can lead to suspicion of sleep hypoxemia - as, for example, obesity and obstructive sleep apnoea with loud snoring and restlessness in sleep, hypoxemia during wakefulness in the overlap syndrome, and nocturnal awakening with wheeze in bronchial asthma. Treatment depends on the cause, and may vary from weight loss and nasal continuous positive airway pressure in obstructive sleep apnoea, to nocturnal oxygen in 'blue bloaters', a combination of these two in the overlap syndrome, and long acting bronchodilators such as slow release theophyllines in nocturnal asthma. Recognition and appropriate treatment of nocturnal hypoxemia is an important new development in respiratory medicine.

2050798098

**FROM MEDLINE {DIALOG: FILE 155}**

**122**

AN- <DIALOG> 08169288
TI- Theophylline and prostaglandin E2 on duodenal bicarbonate secretion: role for 5'-cyclic adenosine monophosphate.
AU- Mu JZ Hogan DL Koss MA Isenberg JI
CS- Department of Medicine, UCSD Medical Center, California.
SO- <JN> Gastroenterology
CP- UNITED STATES
SO- <PY> Jul 1992
SO- <VO> 103 (1) p153-9
SN- 0016-5085
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- Cyclic adenosine monophosphate (cAMP) has been implicated as an intracellular "second" messenger in duodenal mucosal bicarbonate secretion in animals. The purpose of this study was to determine whether cAMP may mediate duodenal mucosal bicarbonate secretion in humans. In healthy volunteers, a 4-cm segment of proximal duodenum was isolated from gastric and pancreaticobiliary secretions. Either the phosphodiesterase inhibitor theophylline, prostaglandin (PG) E2, or a combination thereof was administered topically to the isolated duodenal mucosa. Theophylline (10(-2) mol/L) and PGE2 (10(-5)-10(-4) mol/L) each significantly increased bicarbonate secretion and transmucosal potential difference. Moreover, when theophylline and PGE2 were administered in combination, the duodenal bicarbonate output was additive compared to either agent alone. When theophylline was infused with increasing doses of PGE2, the dose-response curve was shifted to the left. Furthermore, increases in bicarbonate secretion and transmucosal potential difference were correlated significantly. These results suggest that cAMP may act as an intracellular mediator of human duodenal mucosal bicarbonate secretion.

2050798099

**FROM SCISEARCH** {ISI's *Science Citation Index*; DIALOG: File 434}

**123**

GA- FM931
NR- 26
TI- PHARMACOKINETIC-PHARMACODYNAMIC MODELING OF THE INTERACTION BETWEEN FLUMAZENIL AND MIDAZOLAM IN VOLUNTEERS BY APERIODIC EEG ANALYSIS
AU- BREIMER LTM BURM AGL DANHOF M HENNIS PJ VLETTER AA DEVOOGT JWH SPIERDIJK J BOVILL JG
CS- LEIDEN UNIV HOSP,DEPT ANAESTHESIOL,POB 9600/2300 RC LEIDEN//NETHERLANDS/ LEIDEN STATE UNIV,CTR BIOPHARMACEUT SCI,DIV PHARMACOL/2312 AV LEIDEN//NETHERLANDS/
SO- <JN> CLINICAL PHARMACOKINETICS
SO- <PY> 1991
SO- <VO> 20
SO- <IS> N6
SO- <PG> 497-508
LA- ENGLISH
DT- REVIEW
AB- The CNS effects resulting from the combined administration of midazolam and flumazenil were studied in 8 healthy volunteers to develop a model of the pharmacokinetic-pharmacodynamic interaction. Electroencephalograms (EEG) were recorded between Fp1-M1 and Fp2-M2. The EEG parameter total number of waves between 12 and 30Hz (TNW12-30) derived by aperiodic analysis was used to quantify the effect. Following a 15 min baseline EEG recording, infusion of placebo or flumazenil was started. Infusion regimens for flumazenil were designed so that 'steady-state' concentrations of 10 and 20-mu-g/L were obtained. Doses of midazolam 15, 30 and 60mg over 5 min were given 30 min after the start of placebo infusion (session A) or flumazenil infusion to 10-mu-g/L (session B) or 20-mu-g/L (session C), respectively. Venous blood samples were taken until 8h after the start of the flumazenil or placebo infusion. A sigmoid maximum effect (E(max)) model was used to characterise the relationship between the plasma concentration of midazolam which is in equilibrium with the effect compartment concentration (C(e)m) [C(e)m/Kp] and TNW12-30. Within 2 to 5 min of starting the midazolam infusion all subjects fell asleep, with loss of eyelid reflex. They awoke between 25 and 82 min later in all 3 sessions. The mean (+/- SD) plasma drug concentrations of midazolam corresponding to half the maximum increase in TNW12-30 (EC50) were 276 +/- 64, 624 +/- 187 and 1086 +/- 379-mu-g/L in sessions A, B and C, respectively. The half-lives reflecting equilibration between plasma concentration and effect (t1/2k(e)o), estimated by a nonparametric method, were 2.2 +/- 1.2, 3.3 +/- 3.3 and 2.9 +/- 1.2 min for the 3 different sessions. E(max) and N were not affected by flumazenil. In each subject the plot of the average measured steady-state plasma flumazenil concentration versus the EC50 of midazolam showed a linear relationship. The plasma concentration of flumazenil that doubled the EC50 of midazolam (C(f),2) was 6.5 +/- 1.0-mu-g/L. The observed interaction is consistent with the competitive nature of the antagonism of midazolam by flumazenil.

2050298100

10A04

# THEOPHYLLINE, EEG/EP/ERP ETC, & REVS, BK CHAPS, MONOGRAPHS ETC

**FROM MEDLINE {DIALOG: FILE 155}**

**124**

AN- <DIALOG> 08738258
TI- Review: sleep in heart failure.
AU- Yamashiro Y Kryger MH
CS- Department of Respiratory Medicine, University of Manitoba, Winnipeg, Canada.
SO- <JN> Sleep
CP- UNITED STATES
SO- <PY> Sep 1993
SO- <VO> 16 (6) p513-23
SN- 0161-8105
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
RF- 81

**125**

AN- <DIALOG> 08617827
TI- Pharmacokinetic-pharmacodynamic modelling in pre-clinical investigations: principles and perspectives.
AU- Danhof M Mandema JW Hoogerkamp A Mathot RA
CS- Leiden/Amsterdam Center for Drug Research, Division of Pharmacology, University of Leiden, The Netherlands.
SO- <JN> Eur J Drug Metab Pharmacokinet
CP- FRANCE
SO- <PY> Jan-Mar 1993
SO- <VO> 18 (1) p41-7
SN- 0398-7639
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- A new approach to preclinical pharmacodynamic investigations is presented which allows, in addition to information on the nature of the pharmacological properties of new chemical entities, also important quantitative pharmacodynamic information to be derived. A single intravenous dose is administered to chronically instrumented rats and the time course of the pharmacological effect is determined in conjunction with plasma or serum concentrations. Datasets obtained in this way are subjected to simultaneous pharmacokinetic/pharmacodynamic modelling to obtain estimates of pharmacodynamic parameters such as EC50, Emax, Hill factor and the rate of biophase equilibration. The new approach was applied in studies with benzodiazepines, baclofen, antiepileptic drugs and adenosine receptor agonists and antagonists. In these studies quantitative EEG parameters, the threshold for convulsion as determined by direct cortical stimulation and various haemodynamic variables were used as a pharmacodynamic endpoint. For drugs exhibiting agonistic or inverse agonistic properties, realistic estimates of the potency and intrinsic efficacy could be obtained by modelling of the direct effects in the various effect models. Estimates of the potency of competitive antagonists (e.g. flumazenil, cyclopentyl-theophylline) could be obtained on the basis of modelling of the pharmacodynamic interaction with a full agonist. For baclofen finally also the rate of biophase equilibration could be estimated. The results of these studies show that by implementation of pharmacokinetic-pharmacodynamic modelling in pre-clinical investigations, useful quantitative information on the pharmacodynamics of new drugs in vivo can be obtained. It is suggested that this information may be of value in the early phases of pre-clinical drug development and that it may facilitate the subsequent clinical development process.
RF- 17

2050798102

10A04

**126**

AN- <DIALOG> 08382638
TI- Advances in syncope: a combined approach utilizing head-up tilt testing and electrophysiologic evaluation.
AU- Hoch DH Rosenfeld LE
CS- Yale University School of Medicine, New Haven, CT 06510.
SO- <JN> Conn Med
CP- UNITED STATES
SO- <PY> Oct 1992
SO- <VO> 56 (10) p515-23
SN- 0010-6178
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The electrophysiological evaluation of syncope of unknown origin yields a diagnosis in approximately 40% of patients. In the presence of structural heart disease ventricular tachycardia is the most common etiology accounting for 20% of cases. Over the past several years head-up tilt table testing with isoproterenol provocation has highlighted the syndrome of neurocardiogenic syncope. This syndrome accounts for an additional 30-40% of patients with syncope. There is compelling evidence that this syndrome involves the Bezold-Jarisch reflex with excessive stimulation of ventricular mechanoreceptors (C-fibers) located predominantly in the inferoposterior portion of the heart. Tilt table testing is now an established tool both for diagnosis of this syndrome and for guiding therapy with beta blockers, disopyramide, theophylline, or alpha-agonists. Tilt table testing combined with invasive electrophysiological testing significantly increases the diagnostic yield in the evaluation of syncope.
RF- 88

2050298103

**127**

AN- <DIALOG> 07046239
TI- Bioelectrical impedance in clinical practice.
AU- Zarowitz BJ Pilla AM
CS- Department of Pharmacy Services, Henry Ford Hospital, Wayne State University, Detroit, MI 48202.
SO- <JN> DICP
CP- UNITED STATES
SO- <PY> Jul-Aug 1989
SO- <VO> 23 (7-8) p548-55
SN- 1042-9611
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Bioelectrical impedance (BI) relies on the conduction of a low-voltage alternating current through the body. Lean tissue and fluids containing electrolytes conduct the current and cell membranes serve as capacitors and account for capacitive resistance. Fat and bone are poor conductors. Measurement of the voltage drop of the applied current yields resistance (R) and reactance (Xc). R and Xc are used with height, weight, age, and gender in a number of multiple regression relationships to predict body composition compartments such as fat-free mass, lean body mass, extracellular mass, and body cell mass. The technique has been compared with and validated against traditional measures of body composition analysis. In clinical practice, BI has been used to monitor fluid status in burn and dialysis patients, assess changes of body cell mass with nutritional repletion, and predict pharmacokinetic parameters and dose of theophylline and aminoglycoside antibiotics. BI is a noninvasive, safe, rapid, and reproducible technique with exciting potential in clinical practice.
RF- 45

**128**

AN- <DIALOG> 04191978
TI- The use of in vitro brain slices for multidisciplinary studies of synaptic function.
AU- Lynch G Schubert P
SO- <JN> Annu Rev Neurosci
CP- UNITED STATES
SO- <PY> 1980
SO- <VO> 3 p1-22
SN- 0147-006X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 71

**129**

AN- <DIALOG> 03341761
TI- The role of cyclic nucleotides in the CNS.
AU- Phillis JW
SO- <JN> Can J Neurol Sci
CP- CANADA
SO- <PY> Aug 1977
SO- <VO> 4 (3) p151-95
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- On the basis of the information presented in this review, it is difficult to reach any firm decision regarding the role of cyclic AMP (or cyclic GMP) in synaptic transmission in the brain. While it is clear that cyclic nucleotide levels can be altered by the exposure of neural tissues to various neurotransmitters, it would be premature to claim that these nucleotides are, or are not, essential to the transmission process in the pre-or post-synaptic components of the synapse. In future experiments with cyclic AMP it will be necessary to consider more critically whether the extracellularly applied nucleotide merely provides a source of adenosine and is thus activating an extracellularly located adenosine receptor, or whether it is actually reaching the hypothetical sites at which it might act as a second messenger. The application of cyclic AMP by intrcellular injection techniques should minimize this particular problem, although possibly at the expense of new diffulties. Prio blockade of the adenosine receptor with agents such as theophylline or adenine xylofuranoside may also assist in the categorization of responses to extracellularly applied cyclic AMP as being a result either of activation of the adenosine receptor or of some other mechanism. Utimately, the developement of highly specific inhibitor for adenylate cyclase should provide a firm basis from which to draw conclusions about the role of cyclic AMP in synaptic transmission. Similar considerations apply to the action of cyclic GMP and the role of its synthesizing enzyme, guanylate cyclase. The use of phosphodiesterase inhibitors in studies on cyclic nucleotides must also be approached with caution. The diverse actions of many of these compounds, which include calcium mobilization and block of adenosine uptake, could account for many of the results that have been reported in the literature.
RF- 420

**130**

AN- <DIALOG> 02253123
TI- On the mechanism of action of cyclic AMP and its role in synaptic transmission.
AU- Greengard P McAfee DA Kebabian JW
SO- <JN> Adv Cyclic Nucleotide Res
CP- UNITED STATES
SO- <PY> 1972
SO- <VO> 1 p337-55
SN- 0084-5930
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 38

**FROM SCISEARCH** {ISI's *Science Citation Index*; DIALOG: File 434}

**131**

GA- KH130
NR- 169
TI- CARDIAC ELECTROPHYSIOLOGY AND PHARMACOLOGY OF ADENOSINE - BASIC AND CLINICAL ASPECTS
AU- PELLEG A BELARDINELLI L
CS- HAHNEMANN UNIV,MAIL STOP 110,BROAD & VINE/PHILADELPHIA//PA/19102
SO- <JN> CARDIOVASCULAR RESEARCH
SO- <PY> 1993
SO- <VO> 27
SO- <IS> N1
SO- <IS> JAN
SO- <PG> 54-61
SN- 0008-6363
LA- ENGLISH
DT- REVIEW

**132**

GA- HK868
NR- 91
TI- ANTIINFLAMMATORY STRATEGIES FOR THE TREATMENT OF ASTHMA
AU- BARDIN PG JOHNSTON SL HOLGATE ST
CS- SOUTHAMPTON GEN HOSP,IMMUNOPHARMACOL GRP/SOUTHAMPTON SO9 4XY/HANTS/ENGLAND/
SO- <JN> SOUTH AFRICAN MEDICAL JOURNAL
SO- <PY> 1992
SO- <VO> 81
SO- <IS> N6
SO- <IS> MAR 21
SO- <PG> 303-309
LA- ENGLISH
DT- REVIEW
AB- The inflammatory basis of asthma is now beyond dispute and even mild asthmatics exhibit mast cell degranulation, eosinophil infiltration and increases in mononuclear cells in airway mucosal biopsies. The chronic nature of this endobronchial inflammation may cause damage to ciliated epithelium, which, coupled with laying down of cross-linked collagen within the airway wall, leads to partly irreversible airway obstruction. Corticosteroids, which are potent anti-inflammatory agents, decrease bronchial hyperresponsiveness and the clinical manifestations of asthma. Although inhaled corticosteroids produce fewer side-effects, the use of low-dose (10 mg or less) oral treatment may be recommended for patients unable financially to afford inhaled corticosteroids or who are unable to use them effectively. Other anti-inflammatory drugs, including methotrexate, cyclosporin and the newer leukotriene inhibitors, are not yet in general use and may provide new pharmacological approaches to the treatment of asthma in the near future. In all but the mildest asthma, strategies aimed at preventing and decreasing bronchial inflammation should be the primary aim of treatment. The physician should refrain from prescribing only beta-2-agonists to new asthmatics and patients must be educated to increase understanding of the benefits of preventive rather than symptomatic forms of treatment for this chronic disease.

**133**

GA- P5593
NR- 332
TI- DRUG-INTERACTIONS THAT MATTER - A CRITICAL REAPPRAISAL
AU- MCINNES GT BRODIE MJ
CS- UNIV GLASGOW,WESTERN INFIRM,GARDINER INST,DEPT MED,CLIN PHARMACOL UNIT/GLASGOW G11 6NT//SCOTLAND/
SO- <JN> DRUGS
SO- <PY> 1988
SO- <VO> 36
SO- <IS> N1
SO- <PG> 83-110
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050298107

**134**

GA- RA012
NR- 343
TI- A COLLECTION OF THERAPEUTIC, TOXIC AND FATAL BLOOD DRUG CONCENTRATIONS IN MAN
AU- STEAD AH MOFFAT AC
CS- CENT RES ESTAB,HOME OFF FORENS SCI SERV/READINGRG7 4PN/BERKS/ENGLAND/
SO- <JN> HUMAN TOXICOLOGY
SO- <PY> 1983
SO- <VO> 2
SO- <IS> N3
SO- <PG> 437-464
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

# CARBON MONOXIDE, EEG/EP/ERP ETC & HEALTHY/NORMAL SUBJECTS/VOLS

**FROM EMBASE** {*Excerpta Medica*; DIALOG: File 73}

**135**

AN- <DIALOG> 5957699
TI- Apperceptive agnosia due to carbon monoxide poisoning. An interpretation based on critical band masking from disseminated lesions
AU- Campion J. Latto R.
CS- Department of Psychology, University of Liverpool, Liverpool
CS- UNITED KINGDOM
SO- <JN> BEHAV. BRAIN RES.
CP- NETHERLANDS
SO- <PY> 1985
SO- <VO> 15/3 (227-240)
LA- ENGLISH
AB- Apperceptive visual agnosia is normally held to be a specific deficit in 'apperception' - a hypothetical postsensory stage in visual processing. This paper describes the investigation of a patient diagnosed as suffering from a classical apperceptive agnosia resulting from carbon monoxide poisoning. Controlled behavioural testing confirmed the apparent agnosia but revealed that he could be trained to make a number of visual discriminations which had not been apparent from routine clinical examination and that he suffered a number of subtle sensory impairments which likewise had not hitherto been apparent. Evoked potential recording to grating patterns showed a complex pattern of brain responses involving interactions between spatial frequency, orientation and hemisphere recorded from. The data suggested that the agnosia was caused by sensory impairments rather than a deficit in apperception. We proposed that the impairments were caused by loss of certain spatial frequency and orientation information but rejected an interpretation based on the concept of processing channels in favour of one based on object contour masking by a peppery field defect caused by disseminated lesions. This interpretation received some support from fine grain static perimetry, contrast sensitivity function measurement and orientation discrimination in the two hemifields. Qualitatively similar results were obtained in normal subjects whose field was artifically masked. The results have implications for theories of visual agnosia and for theories of vision based on the concept of processing channels.

2050798109

**136**

AN- <DIALOG> 5401117
TI- Moderate carbon monoxide exposure during sleep: neuro- and psychophysiological effects in young and elderly people
AU- Groll Knapp E. Haider M. Jenkner H. et al.
CS- Inst. Environ. Health, Univ. Vienna, A-1095 Vienna
CS- AUSTRIA
SO- <JN> NEUROBEHAV. TOXICOL. TERATOL.
CP- USA
SO- <PY> 1982
SO- <VO> 4/6 (709-716)
LA- ENGLISH
AB- In order to assess age-related effects of carbon monoxide (CO) on brain wave-activity and sleep, auditory evoked potentials (AEP) were measured during sleep in 10 healthy young volunteers (20-25 years) and in a group (N=10) of healthy elderly Ss (55-72 years) under control and exposure conditions in counterbalanced order. Sleep-stages were classified according to the Rechtschaffen and Kales scoring-system. In addition to electrophysiological measures memory-performance and subjective feeling (mood-scale) was assessed before and after sleep. Eight hours of CO-exposure (100 ppm) gave rise to about 8% COHb. In the group of young Ss there was a significant ($p<0.05$) increase of deep sleep (stages 3, 4), and a decrease of stage REM-sleep during CO-exposure, whereas in elderly Ss similar but non-significant sleep-changes occurred. Some CO-related AEP-changes within different sleep-stages, namely shorter latencies in elderly Ss and higher amplitudes in young Ss, approached significance. Within the group of young Ss there was a tendency to more depressed mood after Co-exposure, as well as to Co-related impairment of memory consolidation; no such effects were observed in elderly Ss. The general hypothesis of more pronounced CO-susceptibility of elderly people was, therefore, not supported in this study.

**FROM MEDLINE {DIALOG: FILE 155}**

**137**

AN- <DIALOG> 04669115
TI- Eye movement as an indicator of brain function.
AU- Kojima T Shimazono Y Ichise K Atsumi Y Ando H Ando K
SO- <JN> Folia Psychiatr Neurol Jpn
CP- JAPAN
SO- <PY> 1981
SO- <VO> 35 (4) p425-35
SN- 0015-5721
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- The level of consciousness between the alert and drowsy states was classified into four stages (Alert, Resting I, Resting II, Drowsy) by studying three factors of the EEG patterns on 23 normal subjects. The eye movements recorded by electro-oculograph were divided into two groups, i.e. rapid eye movements (R type, r type) and slow eye movements (S type, s type). The occurrence of each type of eye movements was confirmed to change in close correspondence to the stages of consciousness. The eye movement on 43 cases with a disturbance of consciousness by metabolic disease were recorded longitudinally according to clinical states. The S type movements were predominantly observed in a state of clouding of consciousness, while the R type and R-S type were observed in a delirious state.

## CO & EP/EEG/ERP REVIEWS, BOOK CHAPTERS, MONOGRAPHS, ETC.

**FROM EMBASE** {*Excerpta Medica*; DIALOG: File 73}

**138**

AN- <DIALOG> 842046
TI- Neurobiological and behavioural toxicity in animals. (A review of papers presented at the workshop of PC IAOH sub committee on higher nervous functions, Prague, July 1975)
AU- Baettig K.
CS- Inst. Behav. Sci., ETH, Zurich
CS- SWITZERLAND
SO- <JN> ACTIV.NERV.SUP. (PRAHA)
CP- CZECHOSLOVAKIA
SO- <PY> 1976
SO- <VO> 18/4 (270-274)
LA- ENGLISH
AB- Although the application of behavioral methods in environmental toxicology has not yet reached the same level of sophistication as in the field of psychopharmacology the approach seems nevertheless very promising. Different side effects of environmental substances which otherwise might be overlooked, could be detected by a critical application of such techniques. On the other hand much more research will be needed with respect to the standardization of such methods as well as in view of the interpretation of the obtained results and their comparison with the effects on humans.

**FROM MEDLINE {DIALOG: FILE 155}**

**139**

AN- <DIALOG> 08237370
TI- Cross species extrapolation in neurotoxicology: neurophysiological and neurobehavioral aspects.
AU- Winneke G
CS- Department of Psychophysiology, Heinrich-Heine-Universitat Dusseldorf, F.R.G.
SO- <JN> Neurotoxicology
CP- UNITED STATES
SO- <PY> Spring 1992
SO- <VO> 13 (1) p15-25
SN- 0161-813X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The theory of phylogenetic continuity of animal species is the basis of any comparative or extrapolative endeavour (Calabrese, 1983). Cross species extrapolation is also a prerequisite for hazard identification in general and developmental neurotoxicology. Two steps must be distinguished: The first step is endpoint-based or qualitative, whereas the second is dose-based or quantitative. Comparison of different species, typically rodents, nonhuman primates and humans, in terms of endpoints is preferentially done within a framework of broad functional categories, such as sensory, motivational, cognitive, motor, and social variables. Within each category specific neurobehavioral as well as electrophysiological measures need to be considered; typically the degree of comparability is higher for electrophysiological than for most behavioral measures. For some frequently used behavioral endpoints in human neurotoxicology, such as psychometric IQ, there is no direct animal counterpart. Once the neural substrate of a particular neurotoxic effect has been identified, as is true for several chemicals such as the pyrethroid insecticides, the organophosphates, most nerve gases or MPTP, or if interspecies comparability in terms of endpoints has proven satisfactory, an effort towards quantitative, dose-based extrapolation is needed. Here species-specific differences in toxicokinetics and metabolism must be taken into consideration in order to arrive at valid translations of dose-response contingencies. If at all possible internal rather than external doses should serve as the frame of reference here. Neurotoxic chemicals of environmental concern for which an adequate data base is available for comparative purposes include alcohol, carbon monoxide, lead, methylmercury and polychlorinated biphenyls (PCB). Principles of cross species extrapolation in neurotoxicology will be illustrated by means of representative neurobehavioral and electrophysiological findings.
RF- 52

**140**

AN- <DIALOG> 05739620
TI- Evoked potentials in the assessment of neurotoxicity in humans.
AU- Arezzo JC Simson R Brennan NE
SO- <JN> Neurobehav Toxicol Teratol
CP- UNITED STATES
SO- <PY> Jul-Aug 1985
SO- <VO> 7 (4) p299-304
SN- 0275-1380
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- The present paper reviews strengths and weaknesses of evoked potentials (EPs) as an index of toxic insult to the nervous system. EPs are obtained by averaging successive samples of EEG time-locked to the presentation of stimuli. Components of the resulting waveform can be measured for amplitude, latency, and distribution. Normal ranges of these parameters have been characterized for auditory, visual and somatosensory stimuli. Auditory EPs have been studied in humans exposed to lead, trichloroethylene, and carbon monoxide. Changes in timing of short latency components and in amplitude of later cortical components have been reported. Methyl mercury, n-hexane and carbon monoxide cause complex changes in the waveshape of flash and patterned visual EPs. Similarly, specific components of somatosensory EPs are altered following exposure to carbon monoxide, lead and acrylamide. The current lack of standardized recording and analysis techniques has sometimes generated contradictory results, but the evidence thus far supports the ultimate usefulness of EPs as a neurotoxicological screening tool.
RF- 31

**141**

AN- <DIALOG> 04631940
TI- Physiological aspects of primary hypertension.
AU- Folkow B
SO- <JN> Physiol Rev
CP- UNITED STATES
SO- <PY> Apr 1982
SO- <VO> 62 (2) p347-504
SN- 0031-9333
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 700

**142**

AN- <DIALOG> 03628330
TI- Effect of rapid decompression and associated hypoxic phenomena in euthanasia of animals: a review.
AU- Booth NH
SO- <JN> J Am Vet Med Assoc
CP- UNITED STATES
SO- <PY> Aug 1 1978
SO- <VO> 173 (3) p308-14
SN- 0003-1488
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- Documentation in the literature indicates that death is as painless following the induction of hypoxia by rapid decompression as by other methods that lead to hypoxia, such as exposure to high altitude, carbon monoxide, and inert gases (nitrogen, xenon, and krypton). Many of the signs and symptoms of hypoxia are the same as those for alcoholic intoxication and inert gas narcosis. Moreover, there is good evidence that analogous relationships or mechanisms may exist for hypoxia, inert gas narcosis, and anesthesia.
RF- 60

**143**

AN- <DIALOG> 03136284
TI- Applications of neurophysiological methods in occupational medicine. A review.
AU- Seppalainen AM
SO- <JN> Scand J Work Environ Health
CP- FINLAND
SO- <PY> Mar 1975
SO- <VO> 1 (1) p1-14
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 74

**144**

AN- <DIALOG> 02907391
TI- Effects of moderate CO dose on the central nervous system-- electrophysiological and behaviour data and clinical relevance. pp 217-32.
AU- Haider M Groll-Knapp E Holler H Neuberger M Stidl H
SO- <JN> In: Finkel AJ, Duel WC, ed. Clinical implications of air pollution research. Acton, Mass., Publishing Sciences Group, 1976. WA 754 A514c 1974.
CP- NETHERLANDS
CA- WA 754 A514c 1974
LA- ENGLISH
DT- MONOGRAPH

2050798115

**145**

AN- <DIALOG> 02363633
TI- Neurophysiological effects of hypoxia.
AU- Michael JA
SO- <JN> Monogr Neural Sci
CP- SWITZERLAND
SO- <PY> 1973
SO- <VO> 1 p65-121
SN- 0300-5186
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 216

**146**

AN- <DIALOG> 01644526
TI- Acute poisoning: some myths and misconceptions.
AU- Matthew H
SO- <JN> Br Med J
CP- ENGLAND
SO- <PY> Mar 6 1971
SO- <VO> 1 (5748) p519-22
SN- 0007-1447
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 36

# ACETALDEHYDE, EEG/EP/ERP ETC & HEALTHY/NORMAL SUBJECTS/VOLS

**FROM EMBASE** {*Excerpta Medica*; DIALOG: File 73}

**147**

AN- <DIALOG> 2587778
TI- The calcium carbimide-ethanol interaction: Lack of relation between electroencephalographic response and cerebrospinal fluid acetaldehyde
AU- Hillbom M.E. Lindros K.O. Larsen A.
CS- Res. Lab., State Alcoh. Monopoly, SF-00101 Helsinki 10
CS- FINLAND
SO- <JN> TOXICOL. LETT.
CP- NETHERLANDS
SO- <PY> 1981
SO- <VO> 9/2 (113-119)
LA- ENGLISH
AB- Penetration of acetaldehyde into cerebrospinal fluid (CSF) was studied in healthy human volunteers during calcium carbimide-ethanol interaction. CSF was sampled via lumbar puncture and blood from a cubital vein. CSF and blood acetaldehyde concentrations varied from 1 to 41 and from 22 to 138 mumol/l, respectively. The results indicate that acetaldehyde penetrates the human blood-liquor barrier. Computer analysis of electroencephalograms (EEGs) recorded during the interaction showed reduction in alpha activity with a concomitant increase in delta activity. The changes were similar to those previously observed during 'normal' ethanol intoxication.

**FROM SCISEARCH** {ISI's *Science Citation Index*; DIALOG: File 434}

**148**

GA- NR675
NR- 38
TI- ABSTINENT ALCOHOLICS EXHIBIT AN EXAGGERATED STRESS-RESPONSE TO 2-DEOXY-D-GLUCOSE CHALLENGE
AU- GEORGE DT LINDQUIST T ALIM T FLOOD M ECKARDT MJ LINNOILA M
CS- NIAAA,CLIN STUDIES LAB,BLDG 10,ROOM 3B19,9000 ROCKVILLE PIKE/BETHESDA//MD/20892 NIH,CTR CLIN,DEPT NUTR/BETHESDA//MD/20892 DEPT VET AFFAIRS MED CTR,NATL INST DRUG ABUST,RES UNIT/WASHINGTON//DC/00000
SO- <JN> ALCOHOLISM-CLINICAL AND EXPERIMENTAL RESEARCH
SO- <PY> 1994
SO- <VO> 18
SO- <IS> N3
SO- <IS> JUN
SO- <PG> 685-691
SN- 0145-6008
LA- ENGLISH
DT- ARTICLE
AB- Chronic excessive alcohol consumption can significantly disturb the hypothalamic control of glucose metabolism; however, the mechanism and clinical significance of this disturbance are poorly understood. We used 2-deoxy-D-glucose (2-DG), which produces intracellular glucoprivation, to compare neurochemical, physiological, and behavioral responses to glucoprivic stress between alcoholics abstinent for 3 weeks and healthy volunteers. Twenty-six male alcoholics and 15 male healthy volunteers received intravenous infusions of placebo, 12.5 mg/kg, and 25.0 mg/kg of body weight of 2-DG over 30 min on three separate days, following a random-ordered, double-blind procedure. Minimal effects were observed following administration of the 12.5 mg/kg of body weight dose of 2-DG. Following 25.0 mg/kg, alcoholics showed both exaggerated ACTH and cortisol responses and greater increases in caloric intake when compared with controls. Although anxiety, desire to consume alcohol, plasma progesterone, and sympathetic and adrenal medullary activity all increased following 2-DG, these responses did not differ between alcoholics and controls. The present findings suggest certain specificity for the exaggerated hypothalamic and adrenocortical responses to mild glucoprivic stress in 3-week-abstinent alcoholics.

# ACETALDEHYDE, EEG/EP/ERP REVS, BOOK CHAPTERS, MONOGRAPHS, ETC

**FROM psycINFO {DIALOG: FILE 11}**

**149**

AN- 00472322
TI- Familial alcoholism: A separate entity?
AU- Goodwin, Donald W.
CS- U Kansas Medical Ctr, Kansas City
SO- <JN> Substance & Alcohol Actions/Misuse
SO- 1983 Vol 4(2-3) 129-136
SN- 01918877
LA- English
DT- JOURNAL ARTICLE REVIEW
AB- Discusses evidence indicating that alcoholism runs strongly in families and that the illness may have a genetic component. This evidence has stimulated 2 lines of research. One type involves comparing alcoholics with and without a family history of alcoholism. The other involves comparing college-age sons of alcoholics with sons of nonalcoholics before members of either group have had an extensive drinking history. Studies show that familial alcoholics differ from nonfamilial alcoholics in having an earlier age of onset and symptoms of greater severity. Familial alcoholism has also been associated with a childhood history of hyperactivity and conduct disorder and an adult history of antisocial behavior. Sons of alcoholics compared to controls have been reported to have higher blood acetaldehyde levels after drinking alcohol as well as more subjective and motor tolerance for alcohol. Sons of alcoholics also generate more EEG alpha activity after alcohol and exhibit lower scores on the Categories test of the Halstead-Reitan Neuropsychological Test Battery. About half of hospitalized alcoholics have a family history of alcoholism. Studies indicate that alcoholics with and without a family history of alcoholism differ on a number of variables. Twin, adoption, and high-risk studies add further evidence that ""familial alcoholism'' is a separate diagnostic entity. (29 ref)


**FROM MEDLINE {DIALOG: FILE 155}**

**150**

AN- <DIALOG> 06612744
TI- The role of genetics in the pathogenesis of alcoholism.
AU- Searles JS
SO- <JN> J Abnorm Psychol
CP- UNITED STATES
SO- <PY> May 1988
SO- <VO> 97 (2) p153-67
SN- 0021-843X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
RF- 101

**151**

AN- <DIALOG> 05600398
TI- Studies of populations at high risk for alcoholism.
AU- Schuckit MA
SO- <JN> Psychiatr Dev
CP- ENGLAND
SO- <PY> Spring 1985
SO- <VO> 3 (1) p31-63
SN- 0262-9283
CN- 5526
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- The evidence supporting genetic factors in alcoholism comes from family studies (an alcoholic biological parent is seen in 31 per cent of alcoholics), twin studies (MZ concordance 55 per cent and 28 per cent for DZ twins), and adoption studies (alcoholism 44 per cent higher in adopted out offspring of alcoholics than controls). Once the presence or absence of a biological alcoholic parent is controlled for, rearing experiences and parental loss do not increase the risk for alcoholism. This conclusion justifies the search for genetic factors which might mediate the increased risk, particularly in groups identified as being at high risk for the development of alcoholism. The methodological assets and liabilities of the 'high risk' approach are reviewed, with reference to a detailed discussion of existing longitudinal and cross-sectional studies of high-risk populations. There is little convincing evidence that measurable personality attributes or differences in rate of ethanol breakdown contribute to alcoholism vulnerability, although high risk groups may have a unique EEG pattern in childhood, and in early adulthood decreased intensity of ethanol response, and increased acetaldehyde may be important.
RF- 143

**152**

AN- <DIALOG> 05549070
TI- [Pathogenesis of alcoholic cardiomyopathy]
TI- <Original> Patogenez alkogol'noi kardiomiopatii.
AU- Frolov VA Dvornikov VE
SO- <JN> Patol Fiziol Eksp Ter
CP- USSR
SO- <PY> Jan-Feb 1985
SO- <VO> (1) p62-7
SN- 0031-2991
LA- RUSSIAN
DT- JOURNAL ARTICLE REVIEW
RF- 102

**FROM SCISEARCH** {ISI's *Science Citation Index*; DIALOG: File 434}

**153**

GA- NB225
NR- 35
TI- P300 DIFFERENCES BETWEEN NONALCOHOLIC YOUNG MEN AT AVERAGE AND ABOVE-AVERAGE RISK FOR ALCOHOLISM - EFFECTS OF DISTRACTION AND TASK MODALITY
AU- BAUER L HESSELBROCK VM OCONNOR S ROBERTS L
CS- UNIV CONNECTICUT,CTR HLTH,DEPT PSYCHIAT/FARMINGTON//CT/06030 UNIV CONNECTICUT,SCH MED,ALCOHOL RES CTR,DEPT PSYCHIAT/FARMINGTON//CT/00000 INDIANA UNIV,SCH MED,ALCOHOL RES CTR,DEPT PSYCHIAT/INDIANAPOLIS//IN/00000
SO- <JN> PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY
SO- <PY> 1994
SO- <VO> 18
SO- <IS> N2
SO- <IS> MAR
SO- <PG> 263-277
SN- 0278-5846
LA- ENGLISH
DT- REVIEW
AB- 1. P300 event-related electroencephalographic potentials were recorded from 79 young adult males, cross-classified with respect to the presence/absence of a family history of alcoholism (FHA) and the presence/absence of a personal history of antisocial personality (ASP) disorder. P300s were elicited using visual and auditory oddball tasks. Each oddball task was repeated with a tracking task added as a distracter. 2. In general, distraction increased the latencies and reduced the amplitudes of P300s elicited by the oddball stimuli. The P300 latency increase occurred only in low risk ASP- and FHA- groups. There was no adaptive increase in P300 latency in the higher risk ASP+ and FHA+ groups. 3. Group differences in P300 were restricted to visual tasks. No interpretable group differences in P300 latency or amplitude were found during the auditory tasks.